lungs. From this list we have hospitalized two cases of active disease.

Since September, 1942, we have fluoroscoped 650 high school seniors. Fifteen x-rays were ordered. One case of active tuberculosis was hospitalized and two cases of suspected tuberculosis are now under close observation. We have fluoroscoped 550 adults. Ten x-rays were ordered. One case of active tuberculosis was hospitalized and two cases of suspected tuberculosis are under close observation. The statistics given do not include the 3,000 fluoroscopies done in the Sanatorium clinics. I believe that the persons seen with our portable unit would not have responded had it been necessary for them to come to Santa Rosa. This year we have done 2,502 skin tests on children in the kindergarten, first, fourth, seventh, and ninth grades, and all new students. These do not include any who had positive skin tests in previous years. Out of this group we found 207 positive reactors, all of whom, with as many of their contacts as we could induce to come to the clinics, were fluoroscoped.

RECENT ADMISSIONS TO OAK KNOLL SANATORIUM

Nearly all of our recent admissions to the Sanatorium are men. This is probably due to the fact that when the men, as heads of families, are receiving large wages they are able to provide private care for their dependents. If, on the other hand, the men develop tuberculosis, their income ceases immediately and they are forced to enter the county institution.

At the present time the majority of cases of active tuberculosis are being found from one of the following three sources: (1) Those admitted to the acute hospital and diagnosed after having been admitted for some other complaint. (2) Those persons who voluntarily present themselves at our clinics for examination. (3) Those cases that are picked up by our doctors in private practice. Most of the doctors in our county are now skin-testing and fluoroscoping the patients coming into their offices.

WORK AMONG CIVILIANS

Our immediate problem is how to reach the small groups of employees found in the dairies, chicken ranches, and other minor industries. We plan to purchase a trailer in which we can set up our fluoroscope in a dark room, have some small dressing cubicles, and a desk for a secretary. This trailer could be driven to any one of these plants at any time and the entire personnel examined. This would be done only after the proper groundwork had been laid by the Tuberculosis Association. We plan to go with our fluoroscope into the larger industries as soon as permission can be obtained.

Up to the present we have not been able to satisfactorily solve the problem of the defense workers living in our county and working elsewhere. These persons are scattered throughout every town and many are living in trailer camps. Over and above that, they work very irregular hours. All of this results in a poor response to any voluntary plan for fluoroscoping these persons. I would like to suggest that perhaps the best way for these persons to be surveyed would be in the large industries in their respective counties, following the lead of the Basalt Rock Company. The entire plant personnel could be fluoroscoped. If the results were given officials in the home county of the employees, all contacts and suspicious persons could be followed. There is one loophole in this scheme. Several persons, when told to see their private physician following the Basalt survey, did not appear. When attempts were made to contact them they had already left their jobs and moved out of the county. Probably these persons previously knew they had tuberculosis and, since jobs are plentiful, merely moved to another community in order that their condition might not be suspected. This would occur in a very small percentage of cases.

Finally, there is the question of our transient population who work in the fruit industry for three or four months each year. We are at a loss to know how to properly solve this problem with them.

IN CONCLUSION

The battle against tuberculosis in Sonoma County was started in earnest on January 1, 1938, with the establishment of a full-time health department. At that time the death rate was given as 72 per 100,000. At the end of the fiscal year, 1942, however, our death rate was computed to be 33.7 per 100,000.

Sonoma County Hospital.

THE CYSTIC LUNG*

LOUIS J. RUSCHIN, M. D.
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THERE is no unanimity of opinion regarding the etiologic factor in certain pulmonary cystic structures. This is due to the fact that more than one factor may operate in several instances of pulmonary cystic structures.

An etiologic classification of pulmonary cystic cavernous structures is submitted, but only those disorders about which there is no unanimity of opinion will be discussed, namely, true congenital pulmonary cysts, emphysema, and acute localized emphysematous bullae.

TRUE CONGENITAL CYSTIC LUNG

The greatest variance of opinion exists concerning the occurrence and frequency of congenital cystic lung. Pierce and Dirkse[1] considered true congenital cysts in adults to be rare, and believed that the assumption of the congenital nature was too often founded upon insufficient evidence—due to inadequate antecedent history, and incomplete evaluation of respiratory diseases by the physician.

PATHOGENESIS

Heuter,[2] in 1914, believed that pulmonary cysts developed as a result of stenosis of bronchial

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Read before the California Trudeau Society at Fresno, April 8, 1943.

branches, and he sponsored the term "congenital bronchiectasis" as von Grawitz[3] had in 1880. S. Smith,[4] in 1925, postulated the theory of an embryonic defect in the development of a bronchus. Mueller,[5] in a critical review of the pathogenetic theories prior to 1928, was of the opinion that a bronchiolar bud becomes arrested in its growth before it attains the stage of a hollow tube, and that, at some subsequent date, the terminal part begins to grow and forms a closed sac into which fluid is secreted by the lining epithelial cells.

Simpkins[6] stated that the bronchi develop as small ramifications of entodermal tissue which becomes canalized directly. Crosswell and King,[7] and Harris,[8] believed that an unknown process interferes with this canalization at some point proximal to the termination of that ramification, with the resultant occlusion at that point. Beyond this point canalization continues, and an isolated canalized segment is formed. The mucous membrane of this segment assumes a normal function, and a cyst is formed. The rate of prenatal growth and the size of the cyst at birth depend on the amount of functioning bronchial mucosa entrapped and the capsular strength of the cyst.

Anspach and Wolman[9] believed that the cysts begin as fluid-containing cavities, either single or multiple. If bronchial communication does not occur, the cysts remain fluid-filled. If communication does occur, air will enter the cavity and the degree of expansion will depend upon the character of the communication and the capsular strength of the cyst. Jackson[10] described types of bronchial obstruction represented in mechanics by: (1) stop valve; (2) check valve; and (3) by-pass valve. Autopsy records of Nelson[11] and of Jacobs[12] demonstrated the check-valve type in expansile pulmonary cysts, and the autopsy reports of R. T. Miller[13] and of Hennell[14] revealed the by-pass type of valve in nonexpansile pulmonary cysts.

TABLE 1.—*Classification of Pulmonary Cystic Structures*

I—TRUE CONGENITAL PULMONARY CYSTS.
- Solitary / Multiple — Fluid / Air / Both — Limited expansibility / Expansile (check valve) / Non-expansile (by-pass valve)

II—ACQUIRED CYSTIC STRUCTURES
- a—Aberant germ cells
  - 1. Dermoid and teratoma
- b—Parasitic
  - 1—Hydatid
- c—Degenerative
  - 1—Chronic emphysema — Blebs (interstitial) / Bullae (vesicular)
  - 2—Neoplasms
- d—Inflammatory
  - 1—Bronchiolectatic (honeycomb)
  - 2—Bronchiectatic
  - 3—Pyogenic (epithelialized abscess)
  - 4—Tuberculous (necrosis)
  - 5—Coccidioides
- e—Mixed (inflammatory, developmental, mechanical, degenerative)
  - 1—Acute localized emphysema
  - 2—Tuberculous tension cavities
- f—Developmental
  - 1—Pulmonary hypoplasia

III—NON-RESPIRATORY PULMONARY CYSTIC STRUCTURES
- a—Eventrations and diaphragmatic hernia

PATHOLOGY

The diagnostic confirmation of the condition depends on the histopathologic examination of the cystic structures in the lung. The occurrence of pulmonary cysts in the stillborn and in the newborn, as proved by autopsy, is probably the strongest evidence in favor of the congenital nature of the disorder.[15] Infection may destroy the typical histologic picture.

The number and distribution of the cysts vary a great deal. Schenck,[16] in a survey of 374 cases, found 141 solitary cyst cases, and 233 multiple cyst cases. The survey revealed 21 per cent bilateral cases, 42 per cent right lung involvement, and 37 per cent left lung involvement.

A connection between the cyst and the bronchial tree may be demonstrated. Diamond and Durham[17] noted instances in which lipiodol failed to enter the cystic structures, and suggested that minute openings, or none at all, explain that phenomenon. Noncommunicating cysts are complete in themselves, as demonstrated at autopsy.[3, 18] Stewart, Kennedy, and James,[19] by careful reconstruction of cysts in the wax model, demonstrated that the majority of cysts communicated with the bronchial tree, and that only a small number were true cysts.

The lining of the cysts varies from a high columnar to a cuboidal or flattened epithelium.[18] In the absence of an epithelial lining, a vascular granulation tissue is found.[18, 20] The wall of the cyst lined with epithelium usually shows structures commonly seen in the wall of the bronchi and bronchioles.[21]

The fluid cysts contain a gelatinous, limpid fluid, comparable to egg-white, free of fat, but high in albumin content.[22] Fibrous strands may traverse the cysts, and give the appearance of trabeculations.[21-a]

SYMPTOMS AND DIAGNOSIS

The clinical symptoms vary markedly, and depend largely on the number, size, and location of the cysts, the presence or absence of bronchial communication, the expansile or nonexpansile nature of the cysts, and the presence or absence of infection at the moment, or in the antecedent history. The frequency of the more common symptoms, as compended by Schenck,[16] are: cough, dyspnea, expectoration, cyanosis, fever, hemoptysis, malnutrition, thoracic pain, weakness, anorexia, wheezing, vomiting, palpitation, and epigastric distress. All or part of these symptoms may be present, depending upon the factors outlined above. Also, the clinical symptoms may be nil.[23] Asthmatoid symptoms may be prominent. Emphysema[24] and cor pulmonale contribute their share of the symptomatology. Spontaneous pneumothorax has been recorded in cases of congenital cystic lung.[14, 25]

The diagnosis of congenital pulmonary cysts is not easily established with certainty in the clinical patient. The roentgenogram is usually suggestive. Lipiodol bronchograms are not invariably valuable or conclusive.[17] Bronchoscopy is of little diag-

nostic value except in a negative sense. Physical findings are extremely variable and are not pathognomonic.[18] The diagnosis of congenital pulmonary cysts must be made with proper appreciation of their infrequency in comparison with the acquired varieties.

TREATMENT

The treatment of the true congenital cyst is medical or surgical, depending on the nature of the case. Lung cyst emptying and closing are rare in the literature.[8,25] Asymptomatic fluid cysts have been recorded.[27] Single or multiple, noninfected, nonexpansile cysts in the asymptomatic state may be treated in an hygienic, prophylactic manner. When a multiplicity of cysts involves the lungs bilaterally, surgery usually is impossible. The prevention of the development of an emphysematous state which would further contribute to the symptomatology should be attempted. Should the involvement be unilateral, lobectomy or pneumonectomy is possible. Removal of expansile cysts *in toto* is eminently successful in selected cases.[28,29] Thoracentesis of balloon (expansile) cysts is indicated for the relief of acute symptoms.[8,26-b]

The deflation should not be accomplished in a rapid manner. Cysts of the lung which have become infected will rarely be cured without complete removal or destruction of the secreting lining of the cavity.[15,29] Maier and Haight [30] emphasized the importance of biopsy in cases thought to be chronic empyema.

EMPHYSEMA

That emphysema is a degenerative lesion of the lung is open to debate by the purist. However, it may lead to the formation of large cavernous areas which simulate cysts.

Emphysema may be defined as an overdistention or overinflation of the lung. Overdistention occurs in the inflated lungs during a forceful expiratory effort, when the escape of air is retarded by a closed glottis or bronchial stenotic process. Unequal compression in different portions of the lung allows a local overdistention. Overinflation occurs when portions of the lung are in a nonexpansile condition (fibrotic or atelectatic), and the thoracic cage enlarges to the same degree as in health. The expansile portions of the lung compensate for the nonexpansile by an increase in volume. Overinflation is favored by insufficient expiration, due to loss of lung elasticity and chest-wall elasticity, which accompanies age. The lungs are insufficiently emptied and the inspiratory effort is increased to establish the proper proportion of residual air to tidal air, and to facilitate gaseous exchange. This may progress until the mechanical state of fullest inspiration prevails.

Repeated and prolonged overdistention and overinflation of alveoli lead to destruction of capillary loops in the walls and to atrophy of the walls. Repair and restoration to a normal state are impossible. This local atrophy may progress to the formation of bullae.

W. S. Miller [31] stated that bullae are formed by the distention of the alveoli themselves, and the larger bullae are the result of rupture of the alveolar walls and fusion of the dilated spaces. He stated that blebs are formed by the escape of air into the interstitial tissues of the lung, and localization of air in the areolar connective tissue of the pleura.

Miller suspected that these loculated emphysematous lesions might cause annular shadows in the roentgenogram. Laurell [32] pointed out the importance of localized obstructive emphysema in the production of pulmonary cavities surrounded by annular shadows (of atelectatic lung). Doub [33] reported the first American cases in which autopsy demonstrated pulmonary blebs and bullae in areas corresponding to cavernous annular shadows in the roentgenogram.

TREATMENT

The treatment of emphysema has not been entirely satisfactory. Prophylactic measures are most important when applicable. A relatively small amount of normal lung tissue suffices for the breathing needs of a healthy individual except under stress. Haldane [34] stated that about one-tenth of the total lung tissue will provide for the gaseous exchange needs under resting condition. Emphysema without fibrosis is encountered rarely. Most sufferers with pulmonary emphysema would survive in comparative comfort if their chest could diminish in size to conform to the diminished lung capacity occasioned by fibrosis, instead of having the remaining lung tissue undergo emphysematous change to conform to the chest capacity. Possibly surgical measures with the aid of bronchial spirometry will offer a solution to this problem. Rebreathing exercises, abdominal belts, and pneumoperitoneum have been employed to advantage in selected cases.

ACUTE LOCALIZED EMPHYSEMA

Acute respiratory tract infections are a potent cause of acute localized emphysema. Certain infections exhibit a toxic or trophic disturbance, even distant from the seat of infection, which results in a weakening of the alveolar walls and elastic tissue. Should a severe cough complicate such an infection, the increase in intrapulmonary air pressure facilitates emphysema in these weakened areas. The presence of bronchitic and bronchiolitic inflammatory states may produce a valve action which increases the bullous formation. The inflammatory residua in these bronchi and bronchioles may perpetuate the bullous state long after the acute inflammatory state has disappeared. That bronchi dilate during inspiration, and contract during expiration, has been observed and adequately substantiated. This phenomenon may allow the bronchial inflammatory residua to function as a check valve. Caffey [35] stated that the rapid dilatation and contraction (sometimes followed by reexpansion and recontraction) of pulmonary cystic structures in acute respiratory disorders furnish strong presumptive evidence that inflationary forces are more important than the destructive agents in their genesis. He emphasized the fact that air-fluid levels in pulmonary cavities are not

pathognomonic of lung abscess. Cheney and Garland [36] pointed out that serial roentgenograms of patients recovering from various inflammatory diseases of the lungs, particularly pneumonia, have disclosed an astonishingly large number of bizarre cyst-like lesions. Although most of these lesions remained unchanged, some disappeared spontaneously and some increased in size.

Zarfl,[26-c] in 1933, applied the term *pneumatocele* to this local acute emphysema. Pierce and Dirkse,[1] in 1937, made similar observations, and applied the term *pneumatocele*. Cheney and Garland [36] suggested the term *pneumocele*. The phenomenon is an expression of local acute emphysema.

The above phenomenon has long been observed in tuberculous lungs and contributes no small number of the tension cavities of tuberculous lungs. That emphysematous bullae may be mistaken on the roentgenogram for tuberculous cavities has been demonstrated at autopsy.[37]

The symptomatology of this group of cases is identical with that discussed under congenital cysts. Many of these cystic structures disappear following the subsidence of the precipitating factors. For this reason, the diagnosis of lung abscess should be made only after due consideration of all the anatomical-etiological possibilities.

TREATMENT

Some of these cystic structures persist for months, or apparently permanently. These cases must be individualized in making therapeutic recommendations. The symptomatology, the size, the number, and the reinfection of the locale are determining factors. The main perpetuating factors may frequently reside in the draining bronchiole as a check valve. Complete patency of the bronchiole, or the obliteration of the bronchiole allows for collapse of the bulla, the first by elimination of the check valve which maintains an intrabulla pressure positive in relation to the atmospheric pressure, and the second by absorption of the entrapped air. Transpleural catheter cavernous decompression [38] may be of some value in selected cases after pleural fusion. Bronchoscopic therapeutic approach may prove of value in some instances.

SUMMARY

The etiologic factors and the pathogenesis of congenital cystic lung, chronic emphysematous bulla, and acute localized emphysematous bulla have been discussed. Therapeutic considerations and approaches have been suggested.

A great variety of lung abnormalities and disorders have been lumped together under the one term "cystic lung." While it undoubtedly is true that a number of cases reported are of congenital origin, recent observations seem to indicate that the number is much smaller than was formerly thought. Numerous persons born with apparently normal lungs develop pulmonary cystic cavernous structures later in life. A careful history and repeated examinations over a long period of time often suggest the pathogenesis of acquired pulmonary cavernous structures in individual patients. In at least an equal number, however, exhaustive studies are of no avail, and the etiology remains obscure.

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THE CYSTIC LUNG*

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THE paper of Doctor Ruschin covers the subject of the cystic lung from various standpoints. The different types of cysts, that is, the true congenital, the hydatid cyst, and the acquired cyst, each represent a different type and can be differentiated by a careful history, and particularly by x-ray studies.

The use of iodized oil applied into the bronchus, or directly by a puncture of the chest wall, has furnished considerable information as to the character of the cysts, and also their extent. In the ordinary film of the chest, the fine walls of the cyst are frequently not visualized, but when the films are taken tangentially, complete outlines are frequently observed.

In children, and occasionally in adults, the large balloon cysts are taken for spontaneous pneumothoraces, and the use of iodized oil in this type is particularly valuable.

Another method to determine which is a cyst, and which pneumothorax, is to insert a needle connected with a manometer; if one obtains the usual variations, a little air is introduced into the space. One can then see whether he is in the pleural space or a cyst. I have seen three cases in which iodized oil was put into the pleural space which produced a symphysis of the pleural sheets so that spontaneous pneumothorax could not develop when the cyst ruptures.

Patients may live a long time with cysts, but when they are infected it becomes a surgical problem.

In the emphysematous type, one can see the size of the cyst enlarge from month to month and from year to year. This rarely happens in the congenital type. In one case that I have observed in a man in the eighties with severe dyspnea, the diagnosis of pulmonary cyst was made, when the patient died suddenly, a small carcinoma was found at the end of the bronchus which partially blocked the bronchus and the large cyst developed. At the post-

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